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14. ABSTRACT

We determined the effects of static and dynamic muscle contraction at equivalent workloads on central aortic pressure and wave reflection. At random, 14 healthy men and women (23 ± 5 yr of age) performed a static handgrip forearm contraction [90 s at 30% of maximal voluntary contraction (MVC)], dynamic handgrip contractions (1 contraction/s for 180 s at 30% MVC), and a control trial. During static and dynamic trials, tension-time index was controlled by holding peak tension constant. Measurements of brachial artery blood pressure and the synthesis of a central aortic pressure waveform (by radial artery applanation tonometry and generalized transfer function) were conducted at baseline, during each trial, and during 1 min of postexercise ischemia (PEI). Aortic augmentation index (AI), an index of wave reflection, was calculated from the aortic pressure waveform. AI increased during both static and dynamic trials (static, 5.2 ± 3.1 to 11.8 ± 3.4%; dynamic, 5.8 ± 3.0 to 13.3 ± 3.4%; $P < 0.05$) and further increased during PEI (static, 18.5 ± 3.1%; dynamic, 18.6 ± 2.9%; $P < 0.05$). Peripheral and central systolic and diastolic pressures increased ($P < 0.05$) during both static and dynamic trials and remained elevated during PEI. AI and pressure responses did not differ between

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Wave reflection and central aortic pressure are increased in response to static and dynamic muscle contraction at comparable workloads

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Edwards DG, Mastin CR, Kenefick RW. Wave reflection and central aortic pressure are increased in response to static and dynamic muscle contraction at comparable workloads. *J Appl Physiol* 104: 439–445, 2008. First published December 13, 2007; doi:10.1152/japplphysiol.00541.2007.—We determined the effects of static and dynamic muscle contraction at equivalent workloads on central aortic pressure and wave reflection. At random, 14 healthy men and women (23 ± 5 yr of age) performed a static handgrip forearm contraction [90 s at 30% of maximal voluntary contraction (MVC)], dynamic handgrip contractions (1 contraction/s for 180 s at 30% MVC), and a control trial. During static and dynamic trials, tension-time index was controlled by holding peak tension constant. Measurements of brachial artery blood pressure and the synthesis of a central aortic pressure waveform (by radial artery applanation tonometry and generalized transfer function) were conducted at baseline, during each trial, and during 1 min of postexercise ischemia (PEI). Aortic augmentation index (AI), an index of wave reflection, was calculated from the aortic pressure waveform. AI increased during both static and dynamic trials (static, 5.2 ± 3.1 to 11.8 ± 3.4%; dynamic, 5.8 ± 3.0 to 13.3 ± 3.4%; $P < 0.05$) and further increased during PEI (static, 18.5 ± 3.1%; dynamic, 18.6 ± 2.9%; $P < 0.05$). Peripheral and central systolic and diastolic pressures increased ($P < 0.05$) during both static and dynamic trials and remained elevated during PEI. AI and pressure responses did not differ between static and dynamic trials. Peripheral and central pressures increased similarly during static and dynamic contraction; however, the rise in central systolic pressure during both conditions was augmented by increased wave reflection. The present data suggest that wave reflection is an important determinant of the central blood pressure response during forearm muscle contractions.

tension-time index; exercise pressor reflex; blood pressure

THE PRESSOR RESPONSE to exercise had been thought to be greater as a result of static muscle contraction compared with dynamic muscle contraction; however, initial studies were not performed on the same muscle groups or at equivalent workloads (4, 42). When static and dynamic muscle contractions are performed at equivalent workloads by controlling for the tension-time index [TTI; a measure of muscular force produced over time (2, 35)] by equating peak tension and altering duration, the pressor response has been shown to be the same during static and dynamic contractions (11, 35). Further, measurement of blood pressure during postexercise ischemia (PEI) indicated no difference in metaboreceptor activation of the pressor reflex between conditions (35). Thus it appears that the pressor response to static and dynamic muscle contraction is similar when muscle mass, peak tension, and TTI are held constant. However, to date, studies of the exercise pressor reflex have not assessed central blood pressure, which may provide important information as peripheral systolic blood pressure is not always a reliable estimate of myocardial afterload (23). Systolic and pulse pressures are lower in the aorta than in the arms and legs where they are amplified to varying degrees depending on elasticity and distance to reflection sites (23, 24). Rowell et al. (28) demonstrated that during maximal exercise, central systolic pressure can vary up to 80 mmHg from peripheral systolic pressure. This disparity had been largely ignored until two recent studies confirmed the finding that peripheral systolic pressure overestimates the actual pressure load of the heart during lower body cycling exercise (32, 33).

Central pressure is the pressure that the left ventricle must overcome, thus determining left ventricular workload (24, 38). Central systolic and pulse pressures are clinically important as they have been shown to be markers of disease (6, 26, 37) and predictors of cardiovascular outcomes (27, 30, 41). The central aortic systolic and pulse pressures are determined by the interaction of a forward-traveling wave as a result of left ventricular ejection and the arrival of a reflected wave from the lower body (23). The forward-traveling wave is dependent on the elastic properties of the aorta, whereas the reflected wave, the sum of reflected waves from the periphery, is dependent on the elastic properties of the entire arterial tree, pulse wave velocity (PWV), round trip travel time from the heart to the periphery and back, and the distance to the major reflecting sites (24). Acutely, vasoconstriction can increase arterial stiffness and PWV by increasing mean arterial pressure and by increasing smooth muscle tone in muscular arteries (23). Recently, metaboreceptor activation has been shown to increase stiffness in nonexercising limbs (13). Thus vasoconstriction elicited by the exercise pressor reflex may influence the timing and amplitude of wave reflection. An increase or decrease in the speed and amplitude of reflected waves from the lower body are much more pronounced in the central aorta (23). This is in contrast to measures taken at peripheral sites, such as the brachial or radial arteries, where changes in peripheral systolic or pulse pressure as a result of lower body wave reflection are typically not observed. Therefore, the pressor response to muscle contraction may include alterations in wave reflection and have

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dramatic effects on the central pressure wave that cannot be appreciated with peripheral measures of blood pressure.

The purpose of this study was to determine the effects of static and dynamic muscle contraction performed with the same muscle group, at the same peak tension, and at the same TTI on central pressure and wave reflection at the end of muscle contraction and during 1 min of PEI (to assess metaboreceptor activation of the pressor reflex). We hypothesized that *1*) muscle contraction would result in an early return of reflected pressure waves from the lower body and an augmentation of central aortic systolic pressure; and *2*) the central pressure and wave reflection responses to static and dynamic muscle contraction and to PEI would not differ when contractions were performed with the same muscle group, at the same peak tension, and at the same TTI. Although we hypothesized similar responses to static and dynamic muscle contraction and to PEI on the basis of previous studies of peripheral pressure (11, 35), we included both conditions to ensure we did not miss an unexpected finding.

METHODS

Subjects. Fourteen apparently healthy men and women, assessed by medical history questionnaire, participated in this study (9 male, 5 female; age 23 ± 5 yr; mass 76 ± 14 kg; height 174 ± 9 cm). Subjects were nonsmokers and were asked to refrain from caffeine, alcohol, and exercise for at least 24 h before testing and reported to the lab at least 4 h postprandial. All procedures were reviewed and approved by the Institutional Review Board, and all subjects gave written informed consent. All experiments were carried out in accordance with state and federal guidelines.

Experimental protocol. After a seated resting period of 10 min, subject's brachial blood pressure was taken in duplicate with an automated blood pressure cuff by oscillometric sphygmomanometry (Omron HEM907, Omron Medical). A radial artery waveform was recorded by placing a high-fidelity strain-gauge transducer over the radial artery of the nondominant arm (Millar Instruments, Houston, TX). Subjects then performed two 1- to 2-s maximal forearm contractions with a handgrip dynamometer (Lafayette Instrument, Lafayette, IN), in their dominant arm, to determine their maximal voluntary contraction (MVC). Testing involved three trials: static, dynamic, and control trials. The static and dynamic trials were matched for TTI (integration of tension over time) by altering trial duration so that 90 s of static handgrip contraction at 30% of MVC and 180 s of dynamic handgrip contraction at 30% of MVC performed at 1 Hz were performed. The control trial consisted of 90 s of seated rest and was performed first, followed by the static and dynamic trials in random order. Subjects rested for 20 min following MVC and between each trial to allow heart rate and blood pressure to return to baseline levels. Before (baseline) and during the last 15 s of each trial (end), brachial blood pressure and a radial pressure wave were recorded in the noncontracting arm. A blood pressure cuff was then inflated to 225 mmHg around the distal portion of the upper arm of the contracting side during the last 5 s of muscle contraction. This occlusion was maintained for 1 min postexercise to assess metaboreceptor-induced activation of the exercise pressor reflex. Brachial blood pressure and a radial pressure wave were recorded again during PEI before cuff deflation. Heart rate was monitored throughout by three-lead ECG.

Pulse wave analysis. Applanation tonometry was used to record a radial arterial waveform by placing a high-fidelity strain-gauge transducer over the radial artery (Millar Instruments). Applanation tonometry has previously been shown to record a pressure wave that does not differ from waveforms obtained from intra-arterial measurements (19). The radial waveform was calibrated from the brachial sphygmomanometric measurement of systolic and diastolic pressures because pulse pressure amplification is negligible between these sites (24). A central aortic pressure wave was synthesized from the measured radial artery pressure waveform with the SphygmoCor Px system (AtCor Medical, Sydney, Australia), which uses a transfer function and is Food and Drug Administration approved. The use of a transfer function to approximate the central pressure wave from the radial wave has been validated using both intra-arterially (10, 18, 25) and noninvasively (16) obtained radial pressure waves. The SphygmoCor transfer function has recently been validated for use during exercise (32). Central systolic and pulse pressures derived with this system have shown good agreement with estimates using carotid recordings (1). We chose to record radial waves instead of carotid because they are easier to obtain particularly during exercise. Central pressures and augmentation index (AI) were obtained from the synthesized wave (see Fig. 1). AI is an index of wave reflection and a manifestation of overall systemic arterial stiffness. AI is defined as the ratio of reflected wave amplitude and pulse pressure, or $AI = (P_s - P_i)/(P_s - P_d)$, where P_s is peak systolic pressure, P_d is end-diastolic pressure, and Pi is an inflection point marking the beginning upstroke of the reflected pressure wave. Because AI is influenced by heart rate (40), AI was also normalized to a heart rate of 75 beats/min (AI-75). The travel time (T_R) of the forward wave from the heart to the major reflecting site and back was measured from P_d to P_i. Additional calculations derived from the synthesized aortic pressure wave were the systolic pressure-time index (STI), diastolic pressure-time index (DTI), and subendocardial viability ratio (SEVR). The STI, or area under the systolic portion of the curve, has been shown to be related to systolic load or the work of the heart and oxygen consumption, and DTI, or area under the diastolic portion of the curve, is associated with coronary perfusion (8). The SEVR is the ratio of DTI to STI expressed as a percentage and is an index of subendocardial perfusion (8). Representative radial and central aortic pressure waveforms during control, static, and dynamic trials are presented in Fig. 2.

Statistics. A three × three (condition × time) ANOVA with repeated measures was used to compare differences among the trials. A Newman-Keuls post hoc analysis was used to determine differences within and between conditions. An α-level of $P < 0.05$ level was required for significance, and all data are presented as means ± SE.

RESULTS

All subjects completed each experimental trial. There were no differences in baseline measurements between conditions.

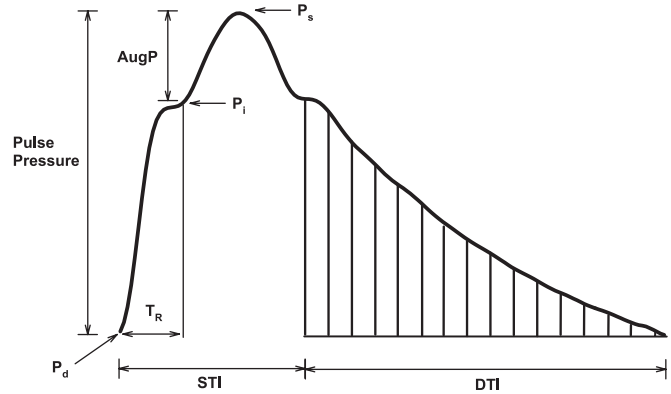

Fig. 1. Variables obtained from the aortic pressure waveform. P_s is peak systolic pressure, P_i is an inflection point that indicates the beginning upstroke of the reflected pressure wave, and P_d is minimum diastolic pressure. AugP is augmented pressure due to wave reflection. T_R is the time delay of the reflected wave or round trip travel time of the forward wave to the major reflecting site and back. Systolic time index (STI) is the area under the curve during systole, and diastolic time index (DTI) is the area under the curve during diastole.

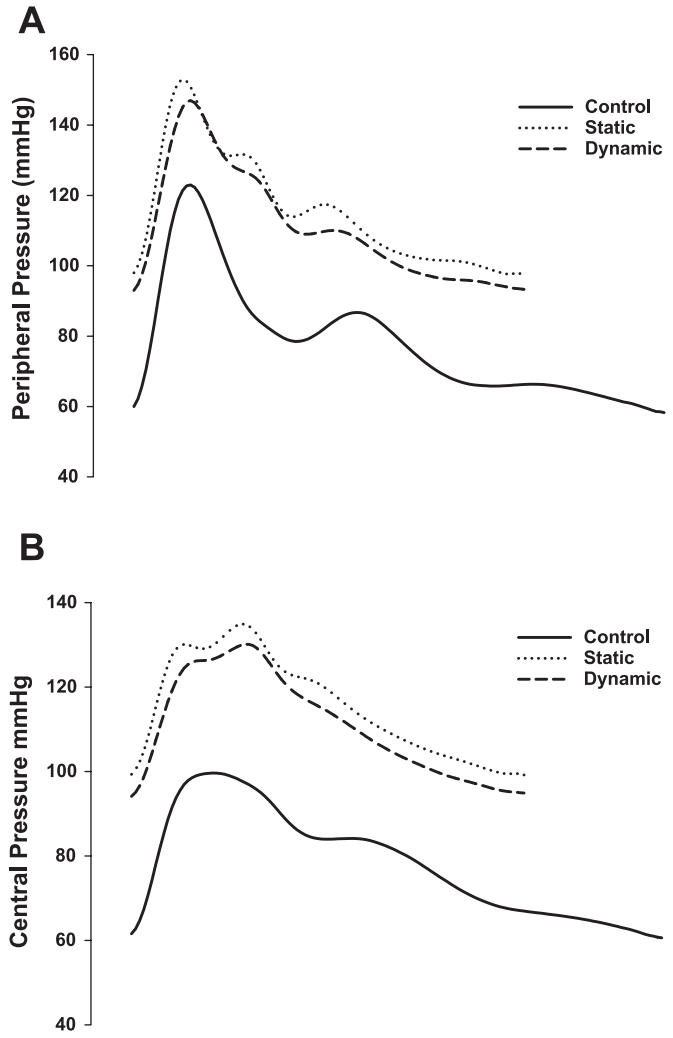

Fig. 2. Representative radial (*A*) and central aortic (*B*) pressure waveforms during control, static, and dynamic trials.

Heart rate was elevated ($P < 0.05$) at the end of both static and dynamic contractions and returned to baseline during PEI (Table 1). At the end of both static and dynamic contractions, peripheral diastolic and systolic pressures did not differ but were increased ($P < 0.05$) compared with baseline and the control condition (Fig. 3*A*). During PEI, peripheral diastolic and systolic pressures remained elevated ($P < 0.05$) for both the static and dynamic conditions (Fig. 3*A*). Central aortic pressures followed a similar pattern. At the end of contraction, central diastolic and systolic pressures increased ($P < 0.05$) at the end of the static and dynamic contractions compared with baseline, and the control condition but did not differ between contraction types (Fig. 3*B*). During PEI central aortic diastolic and systolic pressures remained elevated ($P < 0.05$) for both the static and dynamic contractions (Fig. 3*B*). The magnitude of change in central and peripheral systolic pressure did not differ (Fig. 3*C*). However, there was a greater change in peripheral systolic pressure compared with nonaugmented central systolic pressure (Fig. 3*C*). Nonaugmented central systolic pressure was calculated as central systolic pressure minus the pressure due to the contribution of the reflected wave or the augmented pressure.

AI increased ($P < 0.05$) at the end of both the static and dynamic contractions, but did not differ from each other (Table 1). This response was also significantly different compared with the control condition (Table 1). During PEI, AI continued to increase ($P < 0.05$) in both the static and dynamic conditions (Table 1). AI corrected for a heart rate of 75 beats/min increased ($P < 0.05$) during both static and dynamic conditions but did not increase further during PEI (Table 1). STI increased ($P < 0.05$) at the end of both the static and dynamic contractions, but did not differ from each other (Fig. 4*A*). This response was also significantly different compared with the control condition (Fig. 4*A*). STI remained elevated during PEI in the static and dynamic conditions but was lower than end-contraction measures (Fig. 4*A*). DTI increased ($P < 0.05$) at the end of both the static and dynamic contractions, but did not differ from each other (Fig. 4*B*). DTI remained elevated during PEI in the static and dynamic conditions (Fig. 4*B*). As a result SEVR decreased ($P < 0.05$) at the end of both the static and dynamic contractions, but did not differ from each other (Fig. 4*C*). SEVR returned to baseline levels during PEI in the static and dynamic conditions (Fig. 4*C*).

DISCUSSION

The purpose of this study was to determine the effects of static and dynamic muscle contraction performed with the same muscle group, at the same peak tension, and at the same TTI on central pressure and wave reflection. The primary findings of this study were *1*) peripheral and central pressures were increased similarly in response to static and dynamic muscle contraction and to PEI; *2*) the rise in central systolic pressure during both static and dynamic muscle contraction was augmented by increased wave reflection (as assessed by AI); and *3*) the central pressure and wave reflection response was similar when using the same muscle group, matching peak tension, and holding TTI constant between static and dynamic trials.

Table 1. *Effect of muscle contraction on selected variables*

	Control			Static			Dynamic		
	Baseline	End	PEI	Baseline	End	PEI	Baseline	End	PEI
Heart rate, beats/min	65±2	64±2	65±2	66±3	72±2*	63±2	65±2	75±2*	63±2
AI, %	5.8±3.3	7.0±3.1	9.5±2.5	5.2±3.1	11.8±3.4*	18.5±3.1*†	5.8±3.0	13.3±3.4*	18.6±2.9*†
AI-75, %	0.9±3.2	3.0±3.0	4.6±2.9	1.0±3.2	10.0±3.0*	12.4±2.9*	0.9±3.2	13.3±3.0*	12.8±2.9*
T_R, ms	160±3.9	162±3.5	157±3.9	155±3.9	151±3.6	151±3.9	158±3.9	147±3.5*	147±3.9*
Pulse pressure amplification	1.56±0.04	1.54±0.04	1.54±0.04	1.59±0.04	1.53±0.04*	1.43±0.04*†	1.58±0.04	1.50±0.04*	1.42±0.04*†

Values are mean ± SE. Baseline, before trial; end, during last 15 s of trial; PEI, postexercise ischemia; AI, augmentation index; AI-75, augmentation index normalized for heart rate of 75 beats/min; T_R, time delay of reflected wave. *$P < 0.05$ vs. baseline; †$P < 0.05$ vs. end.

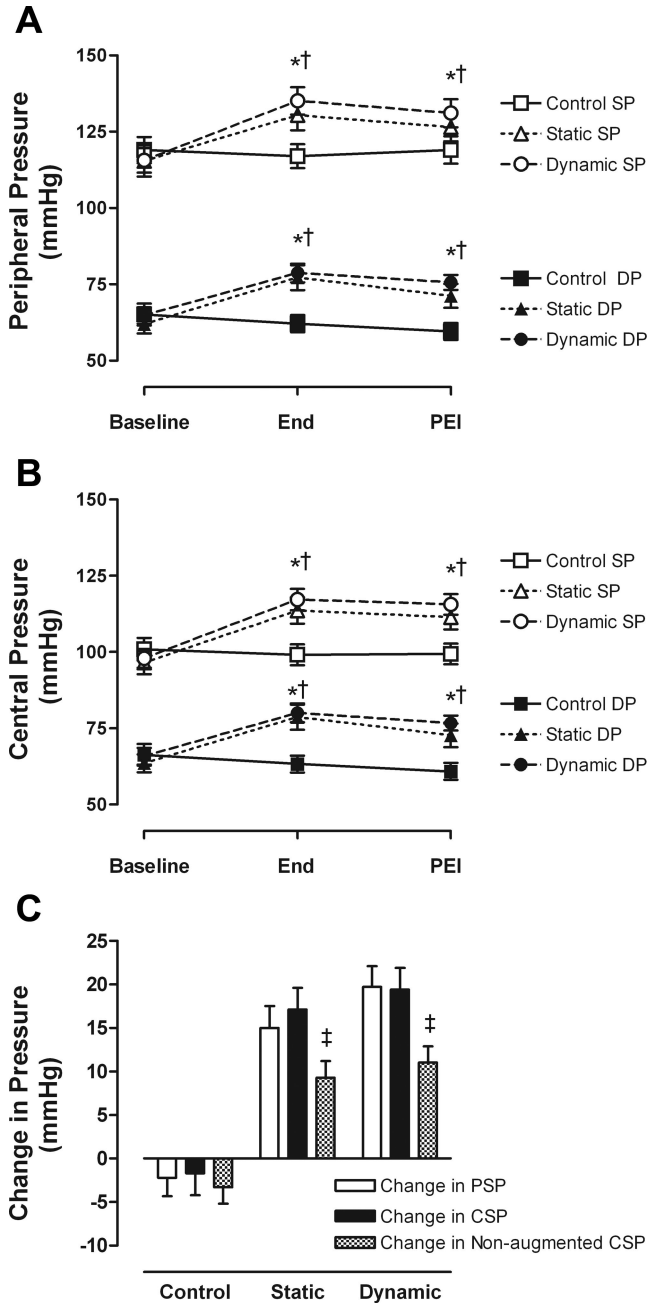

Fig. 3. Peripheral systolic (SP) and diastolic pressure (DP) (*A*) and central SP and DP (B) at baseline, end of each trial, and during postexercise ischemia (PEI) for the control, static, and dynamic trials. Change in peripheral systolic pressure (PSP), central systolic pressure (CSP), and nonaugmented CSP at end contraction (*C*). Values are means ± SE. *$P < 0.05$, dynamic and static vs. baseline. †$P < 0.05$, dynamic and static vs. control. ‡$P < 0.05$ vs. change in PSP and CSP.

Our finding that the peripheral blood pressure response to static and dynamic exercise is equivalent when performed with the same muscle group, at the same peak tension, and at the same TTI is consistent with previous findings in cats and humans (11, 35). We have also demonstrated that increases in wave reflection, as assessed by AI, and central blood pressure do not differ during static and dynamic muscle contraction when performed with the same muscle group, at the same peak tension, and at the same TTI.

The novel finding of our study is that wave reflection significantly augments central systolic blood pressure during forearm static and dynamic muscle contraction. We did not observe differences in the magnitude of change between central and brachial systolic pressure. We did, however, observe a greater change in peripheral systolic pressure compared with nonaugmented central systolic pressure, indicating that wave

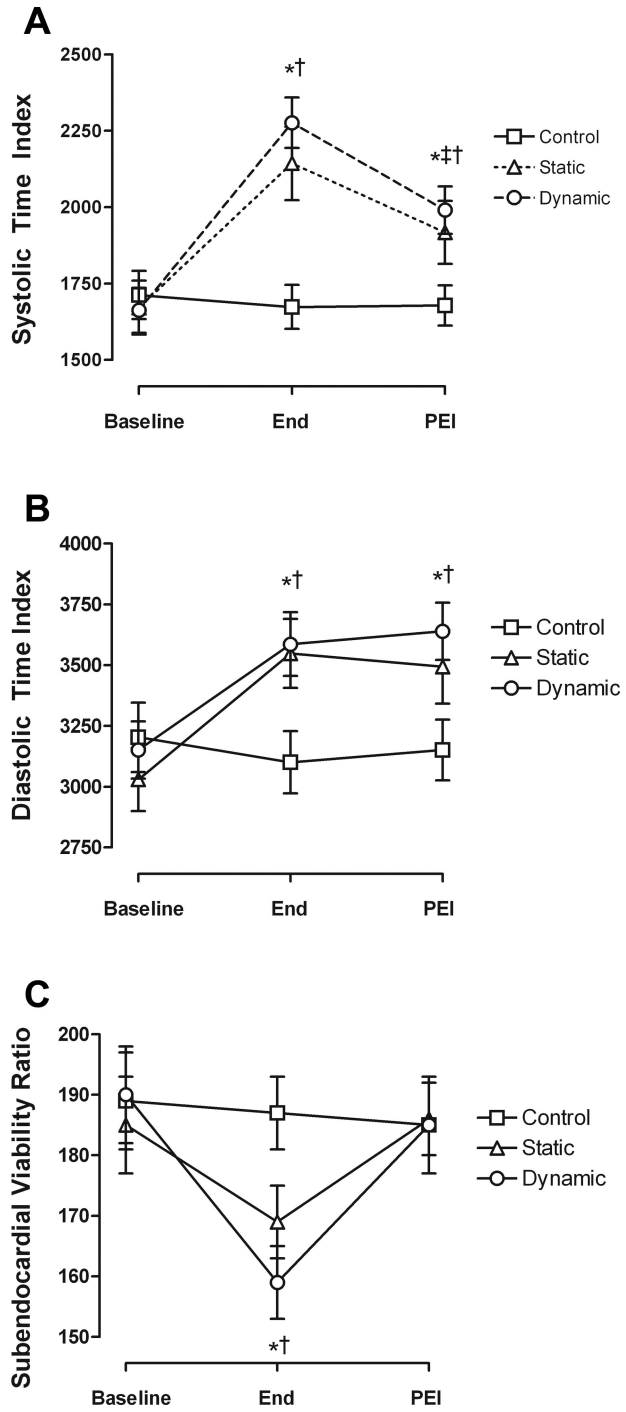

Fig. 4. Systolic time index (*A*), diastolic time index (*B*), and subendocardial viability ratio (*C*) at baseline, end of each trial, and during PEI for the control, static, and dynamic trials. Values are means ± SE. *$P < 0.05$, dynamic and static vs. baseline. ‡$P < 0.05$, static and dynamic vs. end. †$P < 0.05$, dynamic and static vs. control.

reflection is an important determinant of the central blood pressure response to static and dynamic muscle contraction of the forearm.

AI significantly increased during both static and dynamic trials and continued to rise during PEI. Sympathetic activation during muscle contraction could influence wave reflection by increasing PWV through increased mean arterial pressure and vasoconstriction of muscular arteries (23). At higher levels of pressure, wall stress is supported by stiffer collagen fibers as opposed to more compliant elastin fibers at lower pressures (24). Further, sympathetic activation has been shown to reduce compliance in muscular arteries (7), and metaboreceptor activation has been shown to increase stiffness in nonexercising limbs (13). We observed an increase in AI during muscle contraction despite a rise in heart rate, which is inversely related to AI because of a shorter ejection duration (39), suggesting a very robust effect of forearm contraction on wave reflection. The continued rise in AI during PEI can be explained by a drop in heart rate following cessation of muscle contraction in the presence of continued metaboreceptor-mediated sympathetic activation since AI corrected for a heart rate of 75 beats/min did not change from end contraction to PEI.

A limitation of the present study is that we did not measure PWV; however, T_R, the time delay of the reflected wave, is often used as an estimate of aortic PWV. T_R was reduced during dynamic contraction but not static contraction despite a similar increase in wave reflection. It has been demonstrated that AI can change independently of aortic PWV in response to vasoactive drugs (20). Although it is likely that aortic PWV increased similarly during static and dynamic contractions because of similar mean arterial pressure responses, our results suggest the possibility that while the magnitude of wave reflection (AI) may not differ between static and dynamic contraction, the timing of the wave reflection may differ.

Our findings are contrary to the greater increase in peripheral systolic pressure that has been found to occur during lower body cycling and treadmill exercise (28, 33). Sharman et al. (33) demonstrated that upright cycling is associated with a greater increase in peripheral systolic pressure than central systolic pressure due to a progressive decline in AI as exercise intensity increases. The disparate findings can be explained by changes in heart rate and lower body vasodilation in the exercising muscle. AI is inversely related to heart rate (36, 39), which is independent of changes in arterial stiffness (40). Thus the greater increase in heart rate during cycling would be expected to result in a decrease in wave reflection. Vasodilation of the large muscles of the lower body during cycling likely reduce wave reflection (33), whereas we found an increase in wave reflection during forearm contraction when the nonexercising lower body skeletal muscle is likely vasoconstricted (14, 15, 29). Upright cycling also results in an increased in pulse pressure amplification (ratio of peripheral pulse pressure:central pulse pressure) from the central aorta to the periphery (33). We found a small but statistically significant reduction in pulse pressure amplification during both dynamic and static forearm muscle contraction. Taken together this suggests that an equivalent peripheral blood pressure response to forearm and lower body exercise may result in very different central aortic pressures due to differential changes in wave reflection and pulse pressure amplification. It is unclear, however, how central blood pressure would respond to lower body static and dynamic muscle contraction as performed in the present study.

We also examined systolic and diastolic time indexes (STI and DTI) to evaluate the SEVR. STI has been shown to be related to systolic load or the work of the heart and oxygen consumption, and DTI is associated with coronary perfusion (8). The SEVR is the ratio of DTI to STI expressed as a percentage and is an index of subendocardial perfusion (8). STI and DTI both increased during static and dynamic trials; however, the increase in STI was greater, resulting in a decrease in SEVR. Central pressure is a key determinant of left ventricular workload (24, 38) and a predictor of cardiovascular outcomes (27, 30, 41). While the subjects in our study were not at risk for myocardial ischemia, an increase in central systolic pressure and a decrease SEVR during forearm muscle contractions could contribute to myocardial ischemia in patient populations. However, this may be less likely during muscle contractions that elicit increases in DTI as in the present study compared with lower body cycling exercise that results in a drop in DTI with an increase in STI (33).

Previous studies attempting to compare the pressor response to dynamic and static contraction have used dynamic conditions with a lower TTI (12, 21) or equated conditions on whole body oxygen uptake (3, 5), which likely also results in a lower intensity in the dynamic condition due to higher muscle blood flows (35). We used the same handgrip protocol described by Stebbins et al. (35), who demonstrated that peripheral diastolic, systolic and mean pressures were similar in response to static and dynamic contraction performed at 30% MVC and at the same TTI. Rating of perceived exertion in this study was similar between contraction types, indicating a similar activation of central command (35). The blood flow response to dynamic contraction at 30% MVC has been shown to be greater than static contraction at the same intensity and TTI (35), which would be expected to reduce the pressor response by washing out metabolites. To assess the effect of stimulation of the metaboreceptors, we performed 1 min of PEI following each condition. We observed similar peripheral and central pressure responses to PEI, suggesting that accumulation of metabolites that stimulate the exercise pressor response were similar between static and dynamic contraction. Our findings are consistent with previous work equating peak tension and TTI during static and dynamic muscle contraction (35). This may be explained by evidence from both human and animal studies suggesting that dynamic contraction results in greater production of muscle metabolites as a result of greater energy of activation due to repeated contractions (9, 17, 34). It has been proposed that any increase in metabolites during dynamic contraction is offset by their removal by the associated increase in blood flow (35). Our PEI results are consistent with this notion.

We did not include a condition that equated dynamic and static contractions by altering tension instead of time. Previously, Stebbins et al. (35) did not find similar pressor responses between static and dynamic forearm contractions when TTI was matched by equating time and altering tension as opposed to equating tension and altering time. Increasing tension produced during dynamic contractions (60% MVC for 90 s; 1/s) resulted in a greater peripheral blood pressure response compared with static contraction performed at a lower tension

(30% MVC for 90 s). The authors speculate that this is the result of increased dynamic contraction induced production and/or accumulation of metabolites and a greater activation of mechanoreceptors at a higher tension (35).

Although the transfer function used in the present study has previously been validated during exercise compared with invasively determined central pressures (32), our estimates of central pressure may be influenced by any error in the assessment of peripheral pressure at the brachial artery using oscillometric sphygmomanography. The protocol utilized in the present study was of a relatively short duration, and therefore our results may not hold true for progressive static and/or dynamic exercise. The amount of forearm muscle mass involved during handgrip exercise is relatively small and should also be considered when interpreting our results, as the pressor response to static muscle contraction is dependent on muscle mass (22, 31). For example, static contraction of the knee extensors at 30% MVC results in a higher MAP compared with static handgrip contraction also at 30% MVC (31). Therefore, we can speculate that a greater increase in mean arterial pressure during static contraction of a larger muscle mass would be expected to be associated with a greater increase in arterial stiffness and wave reflection. Additionally, reducing the amount of muscle mass during dynamic contractions has been demonstrated to result in hemodynamic changes comparable to static contractions (5). Thus our results may be applicable to activities of daily living that involve short durations and small muscle mass but not to regular endurance exercise involving large muscle groups that has known cardiovascular benefit. Whether static and dynamic contractions, like those performed in the present study, of larger muscle groups would elicit similar increases in central pressure and wave reflection is unknown.

Summary. We have demonstrated that static and dynamic forearm contraction results in an increase in wave reflection and augmentation of central systolic pressure. This is likely attributable to an increase in arterial stiffness as a result of increased mean arterial pressure and vasoconstriction of muscular arteries speeding the wave travel. Our findings are contrary to what has been found during lower body cycling, which is accompanied by a decrease in wave reflection (33). Thus an equivalent peripheral blood pressure response to forearm and lower body exercise may result in very different central aortic pressures due to differential changes in wave reflection, and this should be considered when comparing peripheral blood pressure responses. The present study provides new insight into the blood pressure responses to upper body static and dynamic contraction of a small muscle in healthy young subjects. Future research should be aimed at examining the effect of static and dynamic muscle contraction in other muscle groups (for example, lower body static contraction) on wave reflection and central pressure, in addition to studying the effects in aging and/or coronary artery disease populations to determine clinical significance.

DISCLOSURES

The views, opinions, and/or findings in this report are those of the authors and should not be construed as official U.S. Department of the Army position, policy, or decision unless so designated by other official designation.

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